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CORRESPONDENCE

Extensive subcutaneous fat necrosis after selective head cooling in a newborn with hypoxic ischemic encephalopathy

Dear Editor,

Subcutaneous fat necrosis (SCFN) of the newborn is a rare but self-limited panniculitis developing within few weeks after birth due to several perinatal insults, with perinatal asphyxia being the most common cause.[1,2] The incidence of diffuse SCFN increased after therapeutic hypothermia by whole body cooling introduced for neonatal hypoxic ischemic encephalopathy (HIE), but only one case has been reported in literature after selective head cooling, another method to introduce therapeutic hypothermia in newborn.[3] Herein, we present a case of diffuse SCFN in a newborn with HIE treated by selective head cooling with much more detailed examinations.

A full-term male infant weighing 3320 g was delivered by emergent cesarean section because of fetal distress. His mother received regular prenatal examinations and had experienced an unremarkable pregnancy. Apgar scores were 3 and 6 at 1 minute and 5 minutes, respectively. The infant was resuscitated and intubated immediately, and the laboratory examinations supported the diagnosis of perinatal asphyxia and HIE. Therefore, selective head cooling was performed at 3 hours old. Erythematous patches with progressive hardening developed at 1 day old on the noncooling areas such as cheeks, nape, and back and turned into board-like indurated plaques at 2 days old (Figure 1A). A punch biopsy of a representative lesion from the back at 3 days old confirmed the diagnosis of SCFN, showing subtle but characteristic degenerated adipocytes with radiating needle-shaped clefts (Figures 2A and 2B). In the following days, the hard, erythematous indurated plaques on the back darkened and softened, forming several dusky red, bulging fluctuant cystic nodules (Figure 1B). A magnetic resonance image at 8 days showed liquefaction of the subcutaneous tissue (Figure 2C). Gradual resolution and flattening of the nodules developed (Figure 1C), with concomitant onset of borderline serum hypertriglyceridemia, hypercalcemia, hyperphosphatemia, suppressed intact parathyroid hormone (iPTH), and slightly elevated 1,25-dihydroxyvitamin D3. At 16 days, many subcutaneous well-defined, disc like plaques were noticed on the cheeks and upper arms. Soft tissue sonography showed numerous hyperechoic spots within the plaques, which is compatible with tissue calcification. The fluctuant nodules on the back ruptured at 28 days with some clear oil and whitish creamy discharge (Figures 1D and 1E). Under polarized light, the smear of the creamy discharge showed numerous whitish needle-shaped crystals (Figures 2D and 2E). Under low calcium and vitamin D diet, hydration and diuretics, the serum calcium was maintained within normal range, and renal sonography did not reveal nephrolithiasis at 2 months. Dermatologic follow up at 8 months showed complete resolution of the cystic nodules and no fibrosis or increment of focal vascularity was noticed by soft tissue sonography.

In this report, we describe a newborn developing diffuse SCFN after selective head cooling. Two causal mechanisms have been proposed: (1) tissue hypoxia due to perinatal asphyxia or other insults, and (2) hypothermia-induced crystallization of neonatal fat, which has a high concentration of high melting point saturated fatty acids.[3,4] In our case, in addition to perinatal asphyxia, selective head cooling may still result in moderate hypothermic effects in adjacent areas. The decreasing severity of fat necrosis from the upper to lower back implies a centrifugal effect of hypothermia. Tissue perfusion may be worse on weight bearing areas, and SCFN was reported to spare the anterior trunk,[4] just as in our case. Interestingly, the scalp, where the hypothermic device was directly in contact with, was intact in our case, and it could be due to a better local circulation.

Liquefaction of the necrotic fat, which was prominent in our case, may liberate free fatty acid and triglyceride into circulation resulting in hypertriglyceridemia. Binding of free fatty acid to calcium ions may cause tissue calcification, especially in the setting of necrosis related alkaloid microenvironment and hypercalcemia.[4,5] Since serum hypercalcemia may be life-threatening and develops as late as 6 months after the onset of skin lesions,[6] close observation of serum calcium with a diet control in patients with SCFN was suggested. In conclusion, our case highlights the possible risk of SCFN following therapeutic hypothermia by selective head cooling, and clinical vigilance should be upheld for related complications, including hypercalcemia.

Conflict of interest: The authors declare that they have no financial or non-financial conflicts of interest related to the subject matter or materials discussed in this article.

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Figure 1 Evolution of subcutaneous fat necrosis. (A) 2 days old, selective head cooling with indurated erythematous plaques on the cheeks, nape, shoulders and whole back; (B) 8 days old, formation of fluctuant bulging cystic nodules on the upper back, with (C) resolution at 16 days old; and (D) rupture of the fluctuant cystic nodules with (E) clear oil and whitish creamy discharge from biopsy wound.

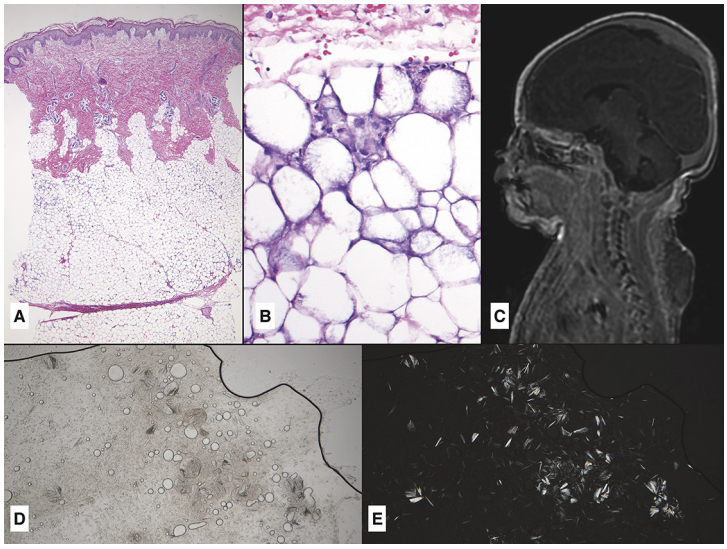

Figure 2 The histopathologic findings from indurated plaques of his left upper back showed (A) a square-shaped punch biopsy with dermal edema and focal fat degeneration [hematoxylin and eosin (H&E) stain, 40×]; (B) radiating strands of cytoplasmic materials with needle-shaped clefts within some degenerated adipocytes (H&E stain, 400×); (C) liquefaction of subcutaneous tissues resulting in cystic nodules on the upper back (MRI); and (D) smear of whitish creamy discharge showed (E) numerous shiny sharp needle-like crystals under polarized light.

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